PLACENTA AND ITS CLINICAL CORRELATIONS

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CERTIFICATE

This is to certify that dissertation entitled “Placenta and its clinical correlations” is the bonafide record of work done by **Dr.M.Elangovan** in the Department of Anatomy, Thanjavur medical college, Thanjavur, during his post graduate course from 2004-2007. This is submitted as partial fulfillment for the requirement of M.S. Degree Examination – Branch V (Anatomy) to be held in March 2007.

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CONTENTS

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INTRODUCTION

The word ‘placenta’ connotes a functional union between fetal and maternal structures, and in the human this union has developed to a remarkable degree, as a consequence of the evolutionary development which has followed internal fertilization of a single ovum and the retention of the embryo within the mother for a relatively long time.

Placenta is a diplomatic intermediary between mother and child; ten months nourisher of the still helpless fetus; the tender supplier of fetal requisites; eminent emissary of fetal waste; the wise interpreter of harmful intruders (to the child); the physiological parasite; siphoning off blood not for itself, but for the growing fetus – which in turn as a token of gratitude carries in it’s body during extrauterine life, the only scar a human being is never ashamed of – “the umbilicus”.

The placenta is the most accurate record of the infant’s prenatal experiences. The fetus, cord and the placenta constitute genetically identical parts of a unit that share the uterine environment.

Physicians, generally are uncomfortable with the task of examining the placenta. Yet it is a task, they should willingly undertake. Submitting this

organ to reasonably knowledgeable look and touch can provide much insight into prenatal life. It helps in caring for the neonate; a record for neonatologist to plan for MCH care, otherwise this valuable information would be discarded along with the organ.

Remarkable advances have occurred in our understanding of normal human embryology, placentation, and fetal growth. In an increasing number of pregnancies, an abnormality may be detected early in gestation, and its etiology determined by investigations using sonographic and genetic analysis.

Knowledge of the human placenta is now reasonably well in hand; not only the gross and light microscopic features. With the noninvasive investigations like sonography, we have entered an era of diagnosing anomalies of placenta even before delivery and there is a scope for interventional procedures to correct them. A placental examination with gross and microscopic studies will yield additional information about the course of the pregnancy.

Hence this study is undertaken with the following aim and objective.

AIM OF STUDY

Placental examination offers a lot of information of prognostic significance for the newborn. This enables the neonatologist to handle the babies much more efficiently.

This prompted me to study the placental shapes, cord insertion, lobes and gestational outcome and to correlatre the morphological / morphometric features with clinical picture.

HISTORICAL PERSPECTIVE

Placenta derives its name from Greek & Latin. The Latin root "Placentos" means a cake; Greek root "Plakios" means flat.

The placenta – a dynamic organ, which is unique in its development has been known to early man. The reason being it was obvious after any childbirth & hence it was called "afterbirth". It was renamed 'Placenta" by Realdus Columbus in 1559.

There are two school of thought about who coined the term. One set of people claim that it was Gabriele de Fallopsis (1523 – 1562) whereas the popular view is that it was Realdus Columbus (1516 – 1559) who named it. Columbus borrowed the Latin term which means "flat cake".

NORMAL ANATOMY

At full term the placenta is disc like, and presents foetal and maternal surfaces, and peripheral margin.

Foetal surface is smooth, covered by amnion and presents the attachment of the umbilical cord close to its centre. Beneath the amnion, umbilical vessels radiate from the cord. Sometimes the extra-embryonic part of the yolk sac, known as the umbilical vesicle is found beneath the foetal surface close to the umbilical cord and is connected by a fibrous remnant of the vitello-intestinal duct.

Maternal surface is rough and irregular, and is mapped out into 15 to 30 polygonal areas known as the cotyledons which are limited by fissures. Each fissure is occupied by a placental septum.

Peripheral margin is continuous with the foetal membrane which consists from outside inwards of fused decidua parietalis and capsularis, chorion leave and amnion.

Measurements – At full term the placenta presents the following measurements :

Diameter – 15 to 20 cm

Thickness – 3 cm (at the centre)

Weight – 500 gms

Proportional weight between placenta and foetus at various stages of pregnancy :

1st month – placenta : foetus = 6:1

4th month – placenta : foetus = 1:1

At birth – placenta : foetus = 1:7

At birth placenta occupies about 30%of the uterine wall.

Structure:

The placenta consists of chorionic plate on the foetal side, basal or decidual plate on the maternal side, stem villi extending between the plates, and the intervillous space between the stem villi filled with the maternal blood.

Chorionic plate is composed of the following structures from within outwards (foetus to mother):

(i) Primary mesoderm containing branches of umbilical vessels (foetal);

(ii) Cytotrophoblast;

(iii) Syncytiotrophoblast;

Basal plate consists from outside inwards of :

(i) stratum spongiosum of decidua basalis;

(ii) outer layer of syncytiotrophoblast (Nitabuch's layer) which undergoes fibrinoid degeneration;

(iii) outer shell of cytotrophoblast;

(iv) inner layer of syncytiotrophoblast; outer zone of this layer also undergoes fibrinoid degeneration and is known as Rohr's Fibrinoid stria.

The basal plate is perforated by the spiral branches of uterine arteries and veins; eventually the intervioous space is filled with maternal blood. Numerous placental septa project from the basal plate into the intervillous space but they fail to reach the chorionic plate. Each placental septum consists of a central core of the decidua basalis containing decidual cells, remnants of endometrial glands and some maternal vessels, and is covered by the cyto- and syncytiotrophoblasts. The areas between the adjacent placental septa are known as the cotyledons which are observed from the maternal surface. Each cotyledon presents in the central axis one or more stem villi. Isolated and multinucleated giant cells are found in the decidua basalis of the

basal plate. The origin of these cells is disrupted, but giant cells are believed to secrete placental hormones.

STEM VILLI connect the chorionic and basal plates, and consist of primary, secondary and tertiary villi with the progress of development.

A primary villus consists of a central core of cytotrophoblast and is covered by the cells of syncytiotrophoblast.

A secondary villus contains a central core of primary mesoderm and is covered successively by cyto- and syncytiotrophoblasts.

A tertiary villus contains in the centre the foetal blood vessels which are surrounded successively from within outwards by primary mesoderm, cyto- and syncytiotrophoblasts. From each tertiary stem villus numerous branching villi project into the intervillous space. The branching villi may fuse with the adjacent villi and the outer and inner walls .of the intervillous space. Therefore, the intervillous space is converted into a sponge-like network of villous type of labyrinthine structure and is filled with maternal blood. The blood vessels within the branching villi do not anastomose with the neighbouring ones. The branching or terminal villi are the functional units of placenta. Each terminal villis is covered by two layers of

trophoblastic epithelium – inner cytotrophoblast and outer syncytiotrophoblast, resting on a basement membrane.

The central core of the villus contains one to six foetal capillaries, and stromal cells. The stroma consists of primitive mesenchymal cells, fibroblasts, collagen fibres, some phagocytic and reniform Hofbauer cells. Syncytial buds invaginate from the trophoblasts and project into the villous stroma.

In early part of pregnancy, about 800 to 1000 villi radiate from the entire chorionic wall. Later, with the regression of chorion leave only 60 stem villi persist in human placenta. Since the number of maternal cotyledons is 15 to 30, it is suggested that each cotyledon contains 2 to 4 major stem villi. A major stem villus and the vessels derived from it form the foetal cotyledon or placentome.

The compartments of the intervillous space are not water-tight, and communicate with one another around the stem villi. The inner wall of the space is formed by chorionic plate and its outer wall by basal plate. The space is lined internally by syncytiotrophoblast, and is filled with maternal blood which never communicates directly with the foetal blood. About 500ml of maternal blood circulates through the intervillous space per minute. Since

the volume of the intervillous space is about 140 ml, the blood within the space is exchanged about four times per minute.

The placental barrier consists of tissues which intervene between foetal blood in the chorionic villi and the maternal blood in the intervillous spacee. Through this barrier exchange of gaseous and metabolic products takes place between the foetus and the mother. Upto the third month of pregnancy the barrier consists of the following four layers from foetus to mother – endothelium of foetal capillaries resting on a basement membrane, a core of primary mesoderm, a basement membrane upon which rest cytotrophoblast and syncytiotrophoblast. From the fourth month onwards, the barrier is practically reduced into two layers – endothelium of the foetal capillaries resting on a basement membrane, and syncytiotrophoblalst which presents numerous micro villi in the intervillous space increasing the total surface area to about 14 sq. metres at full term.

In some areas of terminal villi the syncytium fuses with the stromal capillary wall, forming ‘vasculo-syncytial membrane’. The membranous areas are known as ‘alpha zones’ which are the sites of all materno-foetal exchanges. Rest of the areas of terminal villi are non-membranous and are called ‘beta zones’ where stroma cells and cytotrophoblasts persist. Beta zone possess the sole function of hormone synthesis.

Therefore, human placenta is discoid, deciduate, haemo-chorial, and villous type of labyrinthine organ.

DEVELOPMENT OF PLACENTA :

Placenta is developed from two sources – foetal part from chorion frondosum, and maternal part from decidua basalis.

Stages of development:

When the blastocyst is embedded in the endometrial wall of the uteruss, the endometrium is changed into decidua (due to decidual reaction of stromal cells). The decidua consists of basalis, capsularis, and parietalis. Decidua basalis is situated at the embryonic pole of the blastocyst, the capsularis envelops the rest of the blastocyst and the parietalis lines the uterine cavity. Syncytiotrophoblast proliferates into multilayered and multinucleated protoplasmic mass and advances towards the decidua basalis and capsularis by proteolytic reaction. Meanwhile the cytotrophoblast differentiates internally into a layer of primary mesoderm. Trophoblast and primary mesoderm together form the chorion.

A number of lacunar spaces appear within the syncytiotrophoblast around the entire chorionic wall, but more so towards the decidua basalis.

Each lacuna communicates with the adjacent one around cords of syncytial cells which are known as the trabeculae.

The lacunae enlarge, and erode the branches of uterine arteries and veins. Therefore, lacunae are now filled with maternal blood establishing the utero-placental circulation.

Trabeculae are converted into primary chorionic villi by the invasion of cytotrophoblast in the central axis of each trabecullae. Lacunar spaces are now called the intervillous spaces. At the tips of the primary villi the cells of cytotrophoblast spread outwards and form the outer cytotrophoblastic shell.

The primary mesodermal cells of the chorion invade the central axes of the primary villi and convert them into secondary chorionic villi. The cells of the primary mesoderm extend upto the distal ends of the villi,. And do not form the outer wall of the intervillous space.

Secondary villi are converted into tertiary villi when foetal bnlood vessels derived from umbilical vessels appear within the primary mesoderm. Therefore, all tertiary villi are vascular. From each tertiary villus numerous branching villi project into the intervillous space converting the latter into labyrinthine structure.

The chorionic villi attached to the embryonic pole of the blastocyst proliferate more rapidly, they are called the chorion frondosum. Rest of the chorionic villi attached to the abembryonic pole are known as the chorion leave.

During the 3rd month of pregnancy, the chorion leave degenerate and disappear due tom fusion of decidua capsularis and parietalis.

As a result, the persistent chorion frondosum and decidua basalis form together a human placenta.

CLINICAL CORRELATIONS :

GROSS ABNORMALITIES IN PLACENTATION:

1. PLACENTA EXTRACHORIALIS

In this placenta, the area of the chorionic plate is less than that of the decidual plate and some of the placenta is outside the chorionic plate. Since the superficial decidua is largely spared it appears as a ring or band on the surface of the placenta (placenta marginata) or the membranes may be folded to enclose decidua (placenta circumvallata). Both types may be found on the surface of placenta and may partially or completely encircle the placenta. The condition occurs in about 18 percent of all pregnancies.

2. PLACENTA MEMBRANACEA

The entire fetal membranes are covered with a thin but functioning placenta and the thickness of the placenta is in inverse proportion to its area.

3. PLACENTA SUCCENTURIATA

In this variety, the main placenta is connected to an accessory and small placenta by an artery and a vein which pass through membranes. The clinical significance of placenta succenturiata is that the accessory lobe may be overlooked at delivery and left in utero, and give rise to haemorrhage, infection and the clinical finding of a placental polyp.

4. PLACENTA ACCRETA, INCRETA AND PERCRETA

In a few instances, the trophoblastic villi penetrate deeply into the decidua, or even through the layer and into the muscle without disturbing the gestation. It can only be diagnosed by examining the removed uterus. In such studies portions of the decidua basalis are seen to be absent and the chorionic villi attach directly onto myometrial cells (placenta accreta), penetrate the cells to some degree (placenta increta), or even penetrate through to the serosal surface of the uterus (placenta percreta).

5. HEMANGIOMATA OF THE PLACENTA

These tumours are relatively common, being found in approximately 1 percent of all placentae. Most tumours are small and without clinical

significance but a few are large and are associated with hydramnios, antepartum haemorrhage and premature labour.

6. HYDATIDIFORM MOLE :

A hydatidiform mole is a noninvasive condition in which many of the chorionic villi are characterized by nodular swellings, giving them an appearance almost like bunches of grapes. Commonly, much of the villous surface of the placenta takes on this appearance, in addition the embryo is either absent or not viable. The villi show no evidence of vascularization.

Genetic analysis has determined that hydatiform moles represent the results of paternal imprinting where the female pronucleus of the egg does not participate in development. The chromosomes of hydatidiform moles are paternally derived 46 XX, since the number of lethal genes in 46 YY embryos is not compatible with tissue survival.

7. CHORIOCARCINOMA ;

They are malignant tumors derived from embryonic cytotrophoblast and syncytitrophoblast. These tumors are highly invasive into the maternal decidual tissues and blood vessels. Like hydatidiform moles, most choriocarcinomas, contain only paternally derived chromosomes and are thus products of paternal imprinting.

CLINICAL APPLICATIONS OF PLACENTA

1. CHORIONIC VILLOUS SAMPLING :

A sample of chorion frondosum can be obtained and used for prenatal diagnosis. The procedure is undertaken in trimester 1, after 6 post ovulatory weeks. Usually biopsy forceps of a catheter is inserted through a transcervical route under ultrasonic guidance. Recombinant DNA oprocedures are increasingly being used to identify the molecular defects associated with many heritable diseases. DNA can be obtained either by chorionic villous sampling (trophoblast) or by amniocenteses (and culture of amniocytes). Moreover, DNA can be amplified by the polymerase chain reaction (PCR)

Limb defects have been reported after CVS, the most frequent type being transverse deficiency of either fingers or toes. Vascular disruption following bleeding from the chorion is a likely cause. Another possibility in some instances in inadvertent puncture of the amniotic sac, producing pressure that may perhaps lead to focal necrosis with subsequent repair.

2. AMNIOTIC MEMBRANE

Amniotic Membrane is very thin and transparent and can be used to apply over large areas of burn wound. The role of amniotic membrane in covering partial thickness burn wounds has been well established ever since J. Pigeon (1960) made use of it. Its effective reduction of bacterial count in infected burn wounds was shown by Robson and Krizek (1973) and in contaminated granulation tissue by Bade (1958). It is quite effective due to its covering effect (Morris *et al* 1966) and due to the presence of Lysozyme, Oestrogen and Progesterone (Glask and Snyder 1970). Amniotic membrane alleviates pain, promotes epithelialisation and is also economical (Thompson and Parks 1982; Heberal *et al.*, 1987). Amniotic membrane as an effective burn wound cover for partial thickness burns as has been convincingly noted by Pigeon J (1960), Dino et al (1966), Golocco et al (1974), Bose (1979), Piserchia and Akenzua (1981), Ramakrishnan and Rao (1983), Ramakrishnan et al (1995).

Amniotic membrane is derived from the ectoderm. Amniotic membrane has a basement membrane similar to the one in the wound bed. It consists of type IV collagen, which is essential in healing wounds. The membrane is transparent and has many pores; through which exudation of discharge can take place.

USE OF AMNIOTIC MEMBRANE DRESSING

Earlier, amniotic membrane was used over contaminated wound surfaces and was found that the colony count decreased considerably. Also the amnion and the chorion layers were not separated initially. Later, amnion when separated was found to be very thin and transparent and was advantageous as a temporary wound cover. This thin amnion was often used on superficial partial thickness burn wounds and it is advantageous to use on deep partial thickness burn wounds also in that it prevents infection and bacterial colonization. In larger burn area, when the areas after excision is not suitable for skingrafting, the excised area is covered with amniotic membrane again and this prepares the wound ideally for skingrafting.

Advantage of Amniotic Membrane as a Wound Cover

The following advantages of amniotic membrane as a wound cover are noted:

1. Amnion, when separated is very thin, transparent compared to other wound covers.
2. It is easy to handle the amnion and it is flexible.
3. Amnion is easy to apply, spreads easily and can be used without hurting the patient.
4. Adherence of the membrane to the wound surface is very good and due to its porosity drainage of fluid also takes place.

5. Under the amnion dressing, one could visualize the progress of sound healing, due to its transparency.
6. Pain is much less after the application of the membrane.
7. The large quantity of estrogen, progesterone in the membrane also hastens epithelialisation.
8. When heparin was used as a spray over the burn wound covered with membrane, the healed scar was very satisfactory due to early collagen remodeling. It is easy to combine pharmacological agents like heparin and silver sulphadiazine with the membrane.
9. The cost of burn management and duration of hospital stay was considerably reduced, which is a great advantage in developing countries.

In a developing country with large incidence of burns like in India, amniotic membrane is very useful as a temporary biological dressing.

REVIEW OF LITERATURE

Evolution of knowledge of placenta starts from the Biblical times. Egyptian believed that the placenta was external soul.

The placenta – a dynamic organ, which is unique in its development has been known to early man. The reason being it was obvious after any childbirth & hence it was called “afterbirth”. It was renamed ‘Placenta” by Realdus Columbus in 1559.

There are two school of thought about who coined the term. One set of people claim that it was Gabriele de Fallopsis (1523 – 1562) whereas the popular view is that it was Realdus Columbus (1516 – 1559) who named it. Columbus borrowed the Latin term which means “flat cake”.

According to Henry Gray the normal parameters of placenta are – weight 500 grams (Range 200 – 800 gm), thickness – 23 mm (Range 10 – 40 mm). Thickness is greater at center than periphery.

Hamilton classifies placenta according to the distribution of chorionic villi. They are placenta membranacea [Villi over etire chorionic surface],

zonary placenta [villi grouped in bands around the equator of the chorio all antonic placenta], Bidiscoidal [villi disappear from all except a circumscribed area over the chorion] & placenta succenturiata [accessory lobule].

He also classifies placenta according to the "pattern of distribution of the umbilical vessels after piercing the chorionic plate"

a) The Disperse type [umbilical vessels successively undergo dichotomous division & diminish in caliber]
b) The Magistral type [side branches are given off and reduction in caliber starts in margin. According to Stordania latter type favours fetal growth due to better haemodynamics.
c) Mixed type. – Combination of both the above patterns.

Grosser (1909) provides an acceptable classification of placental morphology, based on maternal & fetal tissue layers interspersed between the fetal and maternal blood streams.

(i) Haemochorial - contains three layers which are trophoblast, connective tissue & endothelium. This is further subdivided into labyrinthine & villous.

(ii) Endothelio chorial – contains four layers, the maternal endothelium being the fourth layer.

(iii) Syndesmo chorial - contains five layers, the endometrial connective tissue being the fifth layer.

(iv) Epithelio chorial – contains six layers, lining endometrium the sixth layer.

Mossman (1937) added one more category to this haemoendothelial – endometrial tissue & trophoblast is destroyed & only the fetal capillary divides the circulation.

Enders suggested the most useful classification of haemochorial placenta depending on the complete layers of trophoblast namely haemomonochorial, haemodichorial & haemotrichorial.

Mossman attributes the name chorio-allantoic to the vascularity homologous to the allantoic vessels of lower animals; haemochorial to the nature of placental membrane ; villous to its villi; decidua to its shedding at birth; discoidal to its circular shape.

The mature placenta consists of two plates, the chorion, and the decidua basalis, between which is the much thicker labyrinth of chorionic villi and intervillous spaces. The cut surface is dark red and spongy. It shows degenerative changes, fibrinoid changes and occasionally cysts.

The fetal placenta is made up of a number of subunits that are now generally known as lobules. The injection studies of Wilkin (1965) have shown that the primary stem villi break up just below the chorionic plate into a number of secondary stem villi which, after running parallel to the chorionic plate for a short distance, divide into a series of tertiary stem villi. The lobules are derived from these tertiary stem villi, which sweep down through the intervillous space to anchor into the basal plate.

The placental septa appear during the third month of gestation : they protrude into the intervillous space from the basal plate and divide the maternal surface of the placenta into 15 – 20 lobes. These septa are simply folds of the basal plate, formed partly as a result of regional variability in placental growth and partly by the pulling up of the basal plate by the anchoring columns, which have a poor growth rate (Boyd & Hamilton 1970).

The human placenta at term weighs around 500 g. It may range between 250-700gm. (das Gupta, Mathur and Gupta, 1974) in Indian subjects. Armitage et al (1967) reported that the average weight of the term placenta was 508gms with extremes of 310gms and 880 gms. There is a positive correlation between the weight of the baby and the placental weight. The human placenta at term averages 1/6 to 1/7 the weight of the foetus.

Strahl classifies placenta into placenta Vera or voll placenta in which the deciduas is in part sloughed off at full term & semi placenta or half placenta – in which the maternal blood space remains intact.

According to Robinson, placentas are of two types – (i) apposed placenta where chorion and the uterine mucosa are apposed and (ii) conjoined placenta where chorion uterine mucosa is fused together.

Whereas, Assheton describes another way of classifying placenta

a) Placenta plicatae – with a simple and unspecialized chorionic epithelium
b) Placenta cumulatae – with highly specialized trophoblast containing lacunae for maternal blood.

The attachments for umbilical cord and vessels to the fetal aspect are variable. According to Boyd, the attachments can be eccentric, marginal and midcentral. Usually the umbilical vessels remain close to each other. Occasionally, they are separated by some distance from the placenta (furcated placenta).

Arey says that the maternal surface shows irregular rough reddish gray lobular areas which correspond to cotyledons.

Langhmann states that cotyledons receive their blood through 80 – 100 spiral arteries that pierce the decidual plate and enter the intervillous spaces at more or less regular intervals. The lumen of the spiral artery is narrow resulting in an increased blood pressure as blood enters intervillous space.

In villi from term placentas the syncytial nuclei are irregularly dispersed and often appear aggregated to form multinucleated protrusions from the villous surface, these being known as syncytial knots. Syncytial knots should be differentiated from syncytial sprouts which are present from the early stages of pregnancy and mainly represent the initial stages in the development of lateral villi.

Placental barrier is not a true barrier according to Arey as many substances pass through freely. At times pathogens gain entry.

As per Langman, the main functions of the placenta are exchange of metabolic and gaseous products between maternal and fetal blood streams and production of hormones.

The clinical significance of placental anomaly has been much debated but it is now clear that the circummarginate form is devoid of clinical importance, and that although circumvallate placentation is associated unduly frequently with a rather small baby, and possibly with a slight excess of congenital malformation (Lademacher et al 1981), it does not appear to be associated with an increased perinatal mortality (Fox 1978). Other aberrant forms of placentation are either of no functional significance, e.g. bilobate placenta, accessory lobe, or so rare that they can, in practical terms, be ignored e.g. placenta membranacea, girdle placenta.

There have been claims that marginal or velamentous insertion of the cord is related to a high perinatal mortality rate but recent studies agreed that the site of cord insertion is unrelated to the incidence of premature labour, low birth weight or fetal hypoxia. (Uyanwah – Akpom & Fox 1977, Woods & Malan 1978). Velamentous insertion does of course present a small risk because of the danger of traumatic damage to, and bleeding from the unprotected vessels running through the membranes.

There appears to be a clear correlation between the presence of a villitis and intra-uterine fetal growth retardation and there has been a tendency

to attribute the deficient fetal growth to damage inflicted on the placenta by the inflammatory processes.

Remarkably little is known about the possible injurious effects on the placenta of toxins, drugs or environmental pollutants. The only example of toxic damage which has been adequately studied is that of the effects of maternal cigarette smoking and low birth weight. The placentae of cigarette smokers show evidence of ischaemic damage which is probably due to vasoconstrictive effects of nicotine on the uterine vasculature (Van der Veen & Fox 1982).

The most important role of the placenta is to transfer oxygen and nutrients from the maternal circulation to the fetal blood. Many have thought that the placenta frequently fails to meet the demands placed upon it and that the resulting condition of placental insufficiency is responsible for many instances of foetal hypoxia, growth retardation or death. In reality, the placenta rarely becomes insufficient for it has a very considerable functional reserve capacity (Fox 1997 a).

Williams et al describe the umbilical cord or the funis as the cord extending from fetal umbilicus to the fetal surface of the placenta. Its exterior is dull white, moist and covered by amnion through which three umbilical

vessels are seen. Its diameter is 0.8 to 2 cms. With an average length of 55 cms. (Range 30 – 100 Cms). Cord length less than 32 Cms is considered abnormally short.

Henry Gray observes that the vessels of the umbilical cord are rarely straight but usually show a twisted configuration. It exists as a right or left handed cylindrical helix. Spiraling occurs in a clockwise (dextral) or anti – clockwise (sinistral) manner.

Arey describes false knots as the blood vessels that are curled up which look like external bulgings.

According to A.L. Mudaliar, placental anomalies can be in the size, form, numbers, attachment of cord and position in uterus.

He classifies diseases of placenta into adherent placenta, placental infarcts, placentitis & tumors of placenta. He also classifies the anomalies of umbilical cord according to its length & insertion apart from knots and torsions of cord.

Teasdale F et al say that placental dysfunction can be evaluated through the quantitative analysis of the morphological changes in the

placental structures that are intimately related to the transfer function of the placenta. He concludes that morphometry is presented as an indirect and non-invasive approach to study the physiology and physiopathology of gestation in the human.

Fox.H describes formation of placenta as under : Trophoblast differentiates into two layer : Outer layer, syncytiotrophoblast and inner layer of cytotrophoblast. A lacunar network develops around the syncytiotrophoblast and fills with maternal blood and endometrial secretions forming primitive uteroplacental circulation. The villi on endometrial side regress leaving smooth surface called chorion leave.

Milovanov A.P.et al suggest a functional system ‘mother – her target organs – uteroplacental area – placenta, fetus and newborn’ to be borne in mind in case of autopsies of maternal deaths.

Emmrich.P. Horn.L.C. and Seifert.U say that definite and possible causes of fetal death and abortion can be due to placental changes such as infection of fetal membranes, disturbances of uteroplacental circulation and placental dysmaturity.

Biagiotti.R.et al report that significant reduction in the proportion of villous tissue occupied by the peripheral villi are consistent with the theory that failure of normal development of terminal villous is responsible for increased vascular resistance in IUGR pregnancies.

Stoz.F., Schumann.R.A. and Schulltz.R. observe that significant differences in placental retardation are between diabetics and control.

Las Heras et al have found that the lumen to whole diameter of fetal arteries is reduced in toxemia of pregnancy.

Jauniaux E et al describe the pathological features of placenta obtained from HIV positive mothers. They include chorioamnionitis and a relative villous hypercellularity.

Because of association of HIV and Non Hodgkin lymphomas, Raphael N. Pollack suggest a careful examination of placenta in a pregnancy complicated by maternal HIV infection to detect any evidence of malignancy.

In this connection, the morphological studies of the chorion & placenta biopsies of mothers aged above 35 years, is important and urgent.

The rest continue to branch and proliferate known as chorion frondosum and forms the bulk of the placenta. Placental septa divide the maternal surface into 15 – 20 lobules that have no physiological significance.

Patten.Z says that the greatest relative size is reached during fifth month, when the placental area is roughly half that of the interior of the uterus.

Pirino.A. et al in their study confirm the existence of single syncytial units joined to constitute the syncytial layer that completely fuse and have functional relationship with the underlying Langhans cells.

Matsubara . S et al have studied the morphology of the mitochondria and endoplasmic reticulum of chorion leave cytotrophoblast and they report that chorion leave cytotrophoblasts afre metabolically active cells as villous and syncytiotrophoblasts have same function relating to fetal membrane physiology.

Potter & Adair have studied the morphology of placenta and estimated the weight of placenta to be roughly about one seventh the weight of fetus.

Hamilton, Mossmann & Boyd claim that placental barrier is thinned nearing them to enable more transmission to meet increased nutritional requirement.

According to Henry Gray the normal parameters of placenta are weight 500 gms, thickness 23mm. Thickness is greater at centre than periphery.

There are multiple shape and forms of human placenta & a variety of types of umbilical cord insertion.

William observes that maternal surface has raised elevated convex areas called lobes – cotyledons separated by grooves of variable depth.

Crawford suggests that the number of cotyledons remains the same throughout gestation. But individual cotyledons grow less actively in the final weeks.

According to Gray & Hamilton, fetal surface is covered by a smooth, shiny transparent chorion. The amnion is closely applied to the chorion on the fetal side and fused with it at the margin.

Crawford observes that the number of trunci as high as 200 although its normal is only 60.

The size, weight and shape of the placenta are all subjected to wide variations.

According to Ramsey, the maternal blood enters the intervillous space in spurts produced by the maternal blood pressure. The blood flows in discrete streams towards the chorionic plate until the pressure is reduced. Lateral spread then occurs.

Placental barrier is a structure of considerable physicochemical complexity, made of four layers, separating maternal and fetal blood. It does not behave like a simple semi permeable membrane.

Getzowa and Sadowsky call it vasculo-syncytial membrane especially around fourth month when it is very thin and there is close association of fetal vessels and the syncytium.

According to Mossmann, the placenta is not a lung, a digestive apparatus, a liver, a kidney nor an endocrine gland. It is an organ in its own right with its own structure which permits to carry out simultaneously the

functions of these organs. In other words, it is analogous to the organs that have been named, but is not in any way homologous with them.

The average length of the normal umbilical cord is between 54 and 61 cm (Fox, 1997) whilst it is thought that a cord length of 32 cm or less should be regarded as abnormally short (Haines and Taylor, 1999). Naeye (1985) has shown that there is a correlation between an unduly short cord and an increased frequency of subsequent childhood mental and motor impairment. Berg and Rayburn (1995) defined an unusually long cord as being more than 80cm whilst Heifetz (1999) regarded a length of 60 cm or more as excessive.

True knots can be formed in the umbilical cord and these are to be distinguished form ‘false knots’, which are either local dilatations of umbilical vessels or focal accumulations of Wharton’s jelly. True knots are found in about 0.5% of all deliveries (McLennan et al., 1988; Heifetz, 1999).

According to Ronald W. Dudek and James D. Fix (2002), the definitive umbilical cord at term is pearl-white, 1 to 2 cm in diameter, 50 to 60 cm long, and eccentrically positioned. It contains the right and left umbilical arteries, left umbilical vein, and mucous connective tissue (Wharton’s jelly).

Chacko and Reynolds state that when fixed in the normally distended state, umbilical arteries exhibit transverse intimal folds called valves of Hoboken.

Teasdale F et al say that placental dysfunction can be evaluated through quantitative analysis of the morphological changes in the placental structures. Morphometry is an indirect and noninvasive approach to the study of physiology and physiopathology of gestation in the human.

The placental parameters probably do not reflect the biochemical activity of the trophoblast say Distler.W et al. They further say that a sufficient capacity of the placenta for estriol synthesis must be taken into account independently of its morphological substrate.

In different conditions complicating pregnancy, placental morphology is deranged.

Aleschchenko I.E. et al state that morphofunctional state of the placenta is altered in hyperthyroidosis in pregnancy.

YinL.Liu states that placental weight and function is reduced as pregnancy becomes postdated.

Keith L. Moore and TVN Persaud (2004) consider placenta as an allograft with respect to the mother. According to them the foetal part of the placenta is a derivative of the conceptus, which inherits both paternal and maternal genes. They postulated theories regarding protection of the placenta from rejection from the mother's immune system. The syncytiotrophoblast of the chorionic villi, although exposed to maternal immune cells within the blood sinusoids, lacks major histocompatibility antigens, and thus do not evoke rejection responses. However, extravillous trophoblast (EVT) cells, which invade the uterine deciduas and its vasculature (spiral arteries), express class I MHC antigens. These antigens include HLA-G, which being nonpolymorphic (class Ib) is poorly recognizable by T lymphocytes as an alloantigen, as well as HLA-C, which being polymorphic (class Ia), is recognizable by T cells. In addition to averting T cells, trophoblast cells must also shield themselves from potential attack by natural killer (NK) lymphocytes and injury inflicted by activation of complement.

Hu L.Lytras et al after their study conclude that the expression of terminal placental differentiation markers such as hGH/CS genes, is altered in term placentas from these diabetics reflecting either impaired placental differentiation or post differentiation impairment of normal placental function.

MATERIALS AND METHODS

A total number of 100 placentae were collected from Raja Mirasudhar Hospital ‘s Maternity ward and operation theatre.

The placentae collected were from normal deliveries & caesarean sections. The collected placentae were washed in tap water and membranes examined. The specimens were transported to the Anatomy Department in 10% Formalin filled bucket.

Following parameters were taken into consideration for the study of placenta & umbilical cord.

a) Weight was measured using a weighing scale.
b) The shape was observed & noted by naked eye examination.
c) The Diameter was measured by a measuring tape,
d) The thickness was measured using a Webers compass.
e) The number of cotyledons were counted visually and
f) The colour of the membranes was noted and presence of any cyst was searched for.

As far as the umbilical cord,

a) The length was measured from the umbilicus of the baby the cut end. The length of cord upto its insertion on the foetal surface of placenta was also measured using a measuring ape. Cord length was calculated by adding both. This was done prior to transporting placenta.
b) The thickness was measured using a measuring tape.

Apart from these morphometric analysis, following were also noted.

(i) Presence or Absence of placental calcification.
(ii) Presence or absence of retroplacental clots.
(iii) Abnormalities of wharton's jelly.
(iv) Abnormalities of umbilical vessels such as single umbilical artery, vessel constriction and segmental thinning were looked for
(v) Type of insertion of umbilical cord was noted.
(vi) Presence of true knots and false knots were looked for as clinical correlation was made.

Details from case sheets were noted which included age, parity & complication of any such as pregnancy induced hypertension, gestational Diabetes, Anemia and Rh In-compatibility.

The following particulars of the babies were obtained, viz

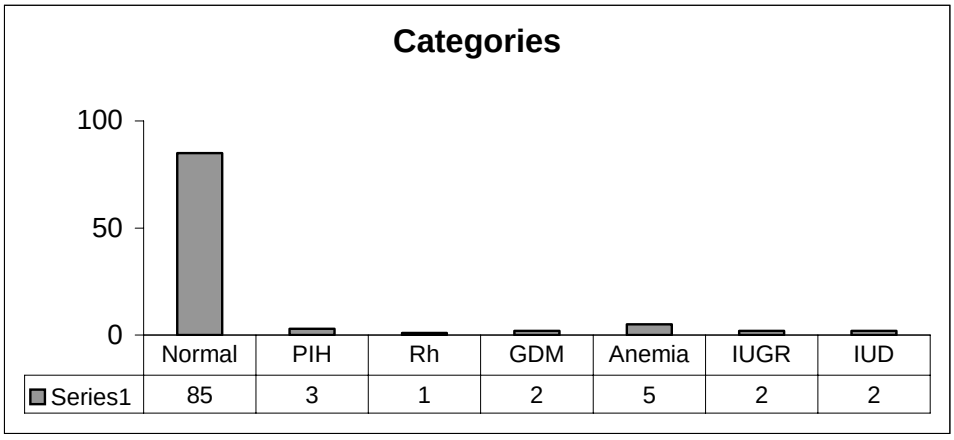
a) Sex b) Weight

OBSERVATION

Of the 100 placentae collected, 53 were from primi and 47 were from multi gravida. 85 were from uncomplicated pregnancies and 15 were from complicated pregnancies. The 15 cases included pregnancy induced hypertension 3, anemia 5, Gestational Diabetes mellitus 2, Rh incompatability 1, IUGR 2 and IUD 2.

Table No. 1.

CATEGORIES	NO. OF PLACENTA	SPECIMEN NO.
Normal	85	1, 2, 3, 5, 6…
PIH	3	12, 22, 34
Rh incompatability	1	4
GDM	2	7, 18
Anemia	5	55, 72, 75, 76, 77
IUGR	2	47, 49
IUD	2	37, 52



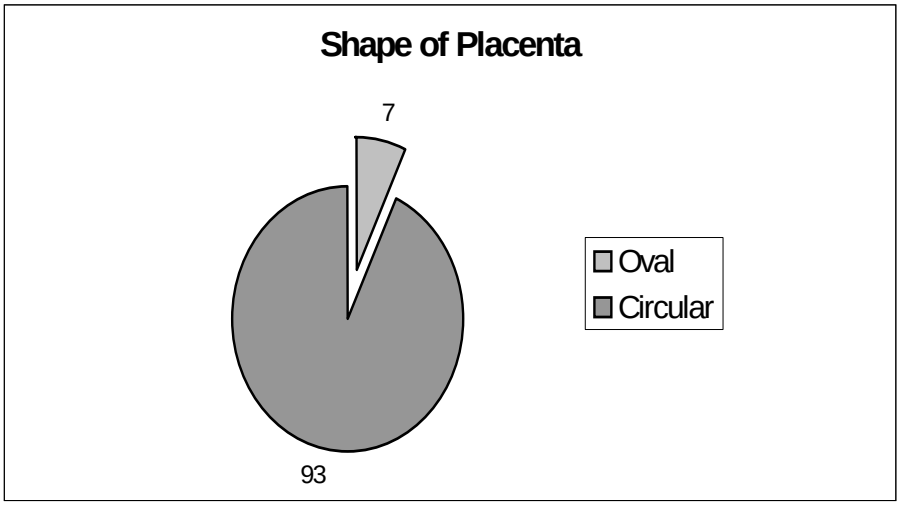
MORPHOLOGICAL PARAMETERS OF PLACENTA

a) SHAPE

Out of 100 specimens 93 were circular and 7 were oval in shape.

Table No. 2.

SHAPE	CIRCULAR	OVAL
No. of Placenta	93	7
Specimen No.	1, 2, 3, 4, 5…	9, 25, 36, 47, 52, 66, 87



b) DIAMETER & THICKNESS :

In the present study, the range of the placental diameter was from 14 cms to 22 cms, average being 17.5.

The range of the placental thickness was from 1.1 cms to 2.5 cms, average being 1.8 cms.

Table No. 3.

DIAMETER (cm)	NO. OF PLACENTA	SPECIMEN NO.
14.1-16	32	2, 3, 4, 7, 8…
16.1-18	34	1, 5, 6, 10, 11…
18.1-20	26	9, 14, 20, 26, 29…
20.1-22	8	38, 55, 61, 68, 74…

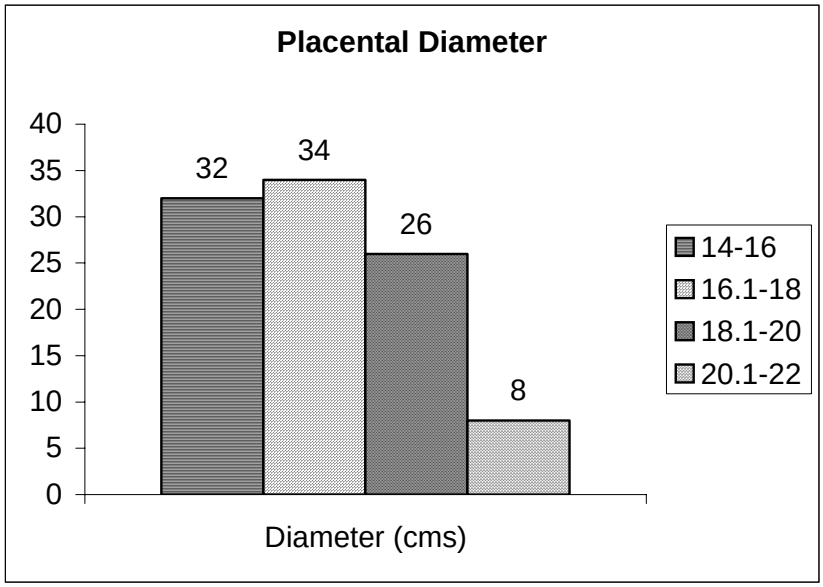
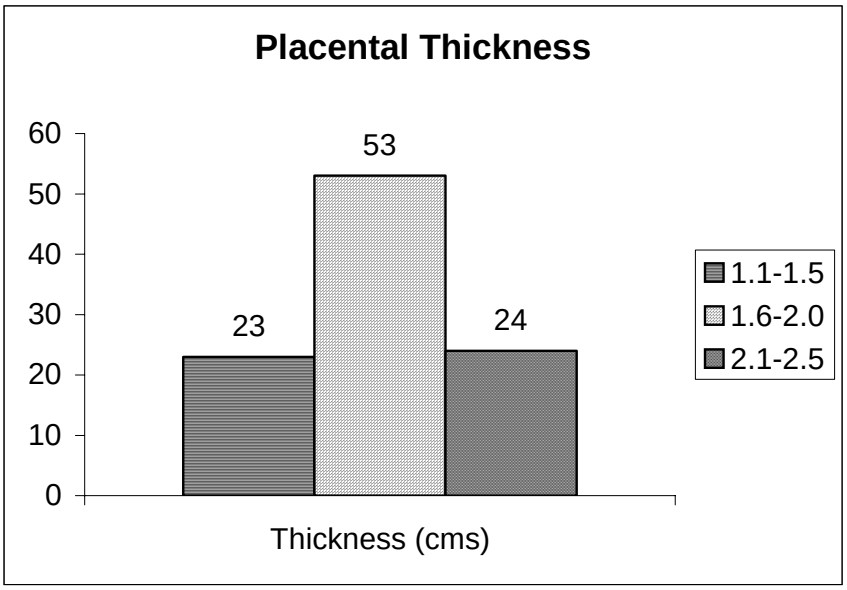


Table No. 4.

THICKNESS (cm)	NO. OF PLACENTA	SPECIMEN NO.
1.1-1.5	23	1, 2, 6, 13, 23…
1.6-2.0	53	3, 9, 10, 11, 12…
2.1-2.5	24	4, 5, 7, 8, 15…



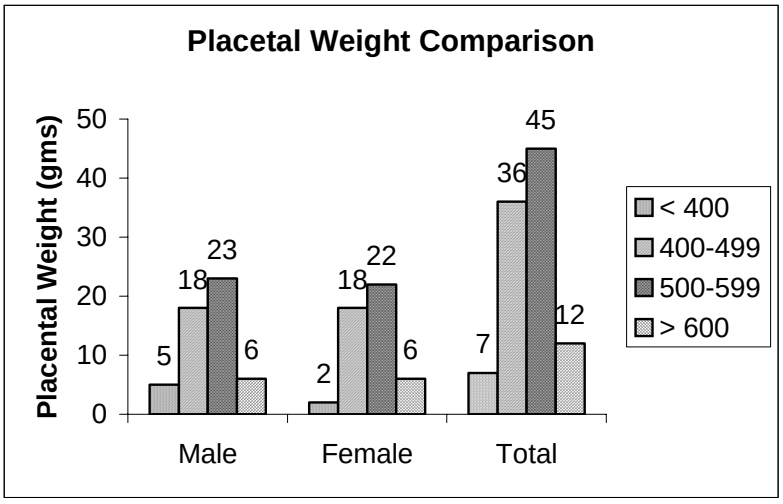
c) WEIGHT :

The minimum weight of placenta in case of male baby was 250 gms and maximum was 760 gms with an average of 504.8 gms.

The minimum weight of placenta in case of female baby was 230 gms and the maximum was 680 gms with an average of 512.7 gms.

Table No. 5.

Placental Wt. (gms)	Male	Female	Total No. of Placenta
< 400	5	2	7
400 – 499	18	18	36
500 – 599	23	22	45
> 600	6	6	12

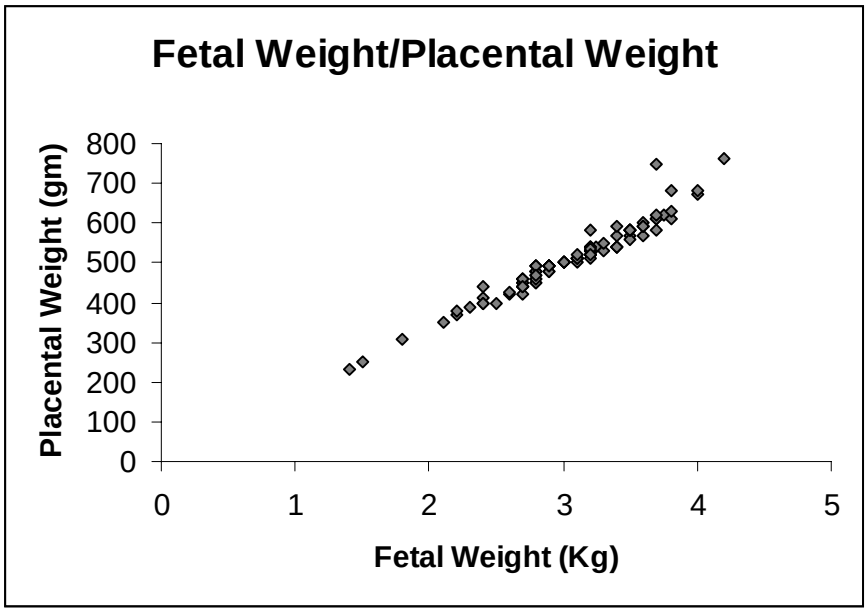


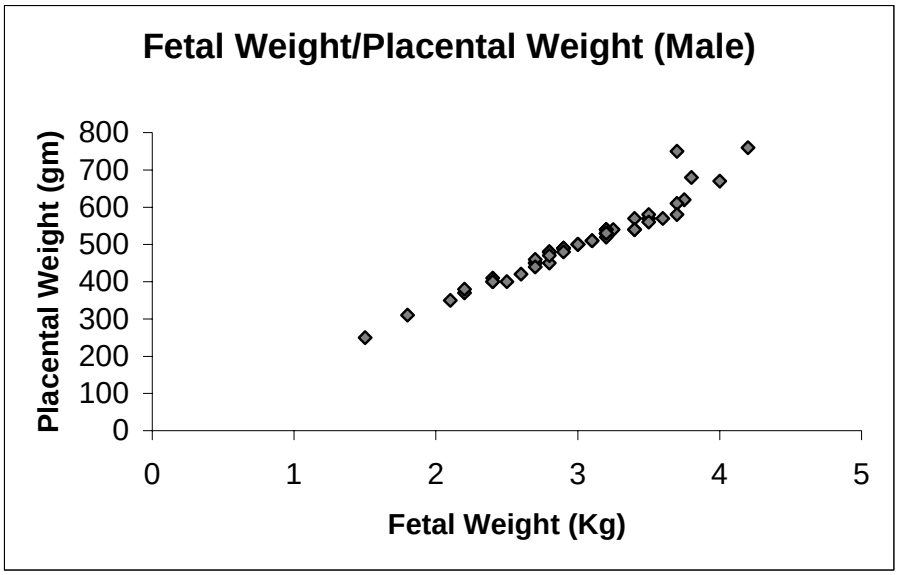
d) FOETO-PLACENTAL RATIO :

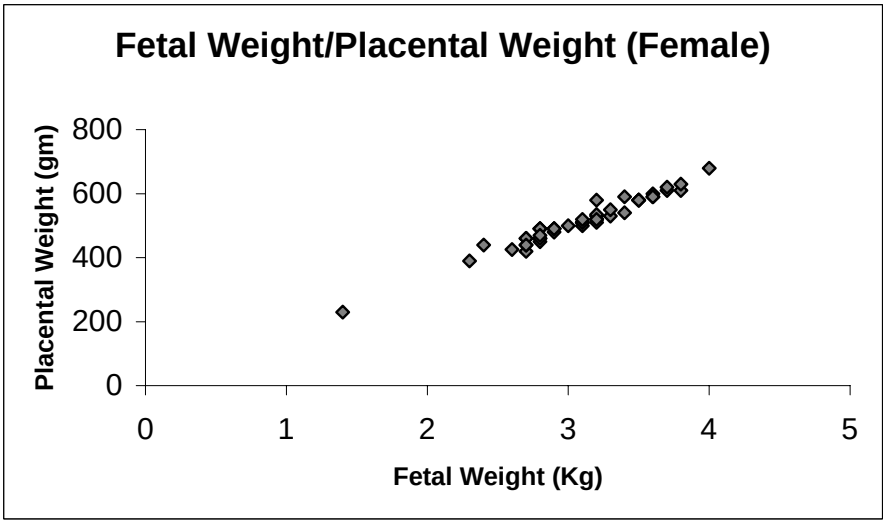
It is the ratio of foetal weight to placental weight. It is normally 6:1 according to Hamilton & Boyd. In the present study it was 5.98:1 in case of male babies and 6.02:1 in case of female babies.

Table No. 6.

	Foetal Wt (kg)	**Placental Wt (gms)**	**Foeto Placental ratio**
Male	3.017	504.81	5.98
Female	3.083	512.71	6.02




Fetal Weight/Placental Weight (Male)
Placental Weight (gm)
800
700
600
500
400
300
200
100
0
0
1
2
3
4
5
Fetal Weight (Kg)


Fetal Weight/Placental Weight (Female)
Placental Weight (gm)
800
600
400
200
0
0
1
2
3
4
5
Fetal Weight (Kg)

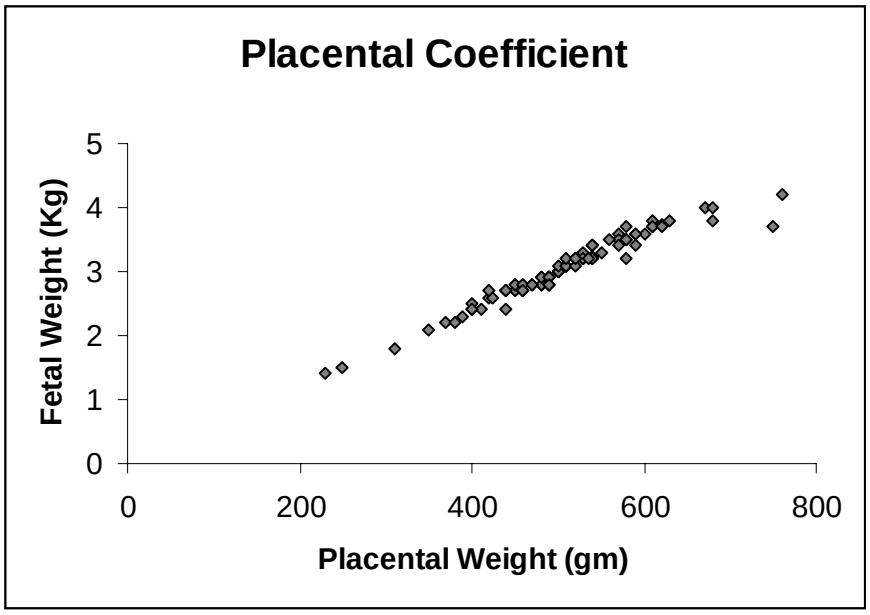
e) PLACENTAL COEFFICIENT :

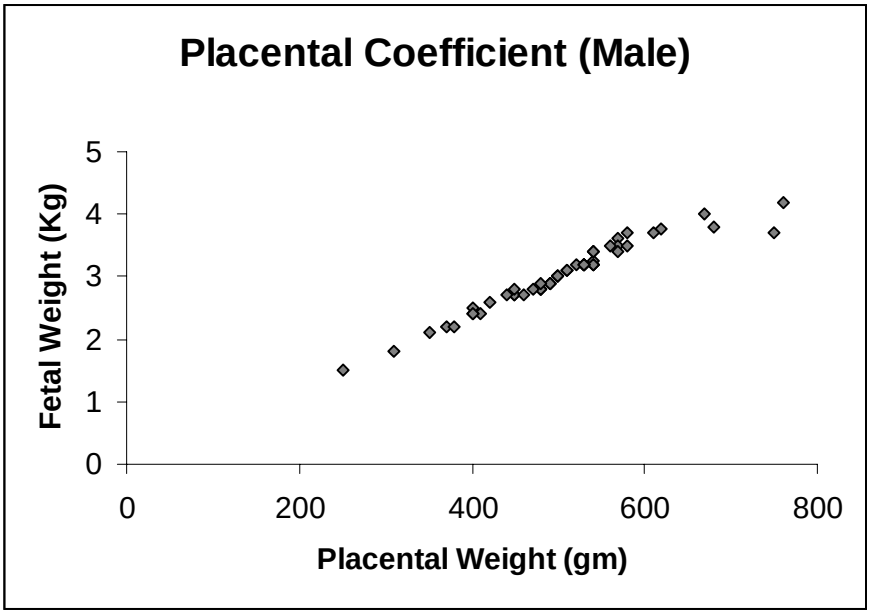
It is ratio of placental weight to foetal weight. It is 0.15 normally, according to Little.

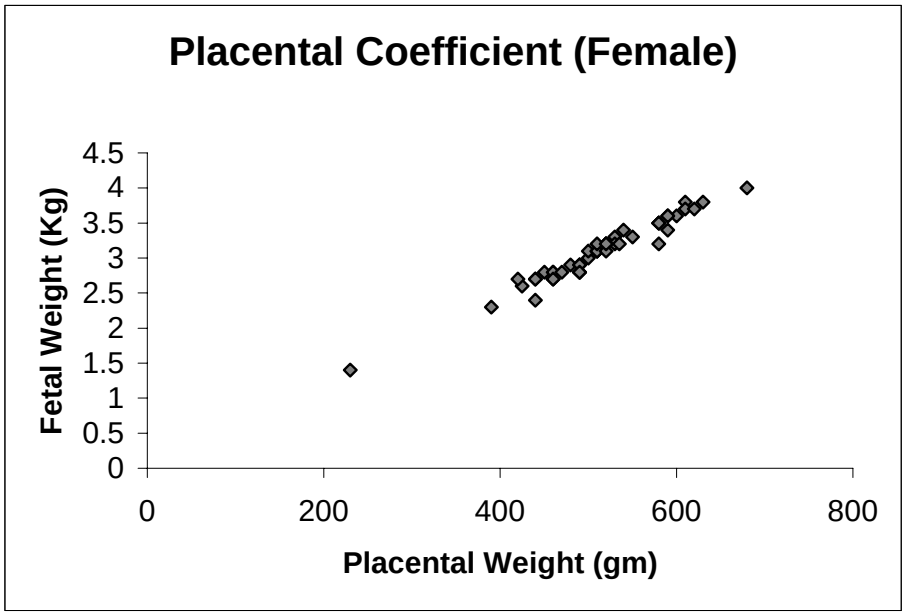
In the present study, the placental coefficient in male babies was 0.17 and in case of female babies was also 0.17.

Table No. 7.

	Foetal Wt (kg)	**Placental Wt (gms)**	**Placental Coefficient**
Male	3.017	504.81	0.17
Female	3.083	512.71	0.17




Placental Coefficient (Male)
Fetal Weight (Kg)
0
1
2
3
4
5
0
200
400
600
800
Placental Weight (gm)


Placental Coefficient (Female)
Fetal Weight (Kg)
0
0.5
1
1.5
2
2.5
3
3.5
4
4.5
0
200
400
600
800
Placental Weight (gm)

f) PLACENTAL WEIGHT IN VARIOUS FACTORS COMPLICATING PREGNANCY :

The average placental weight in case of gestational diabetes was 755 gms. In case of anemia complicating pregnancy, it was 446 gms. It Pregnancy Induced Hypertension, it was 393 gms.

In case of gestational diabetes, the fetoplacental ratio was 5.2 whereas placental coefficient was 0.19.

In case of anemia complicating pregnancy, fetoplacental ratio was 5.8 and placental coefficient was 0.17.

In case of PIH, fetoplacental ratio was 5.8 and Placental coefficient was 0.17.

Table No. 8.

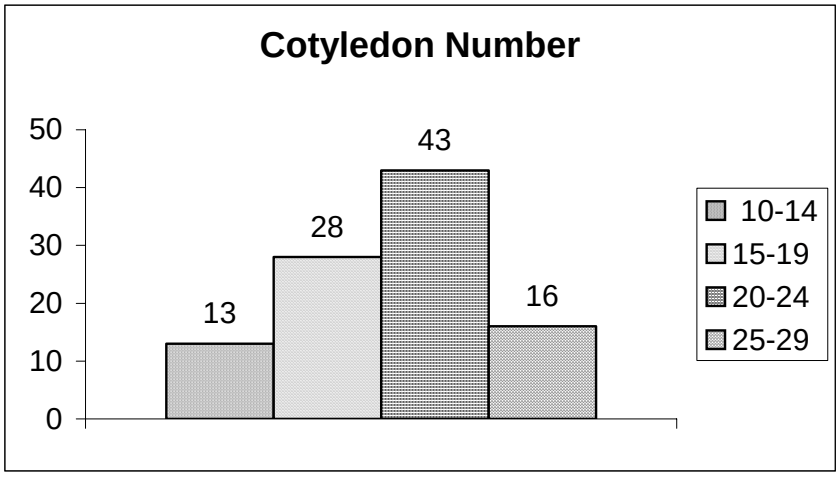
	Average Placental Wt (gms)	**Specimen No.**
GDM	755	7, 18
PIH	393	12, 22, 34
Anemia	446	55, 72, 75, 76, 77

g) NUMBER OF COTYLEDONS :

The number of cotyledons in normal pregnancy ranged from 10 - 29, average being 20. In cases of pregnancy induced hypertension, it was 18. In gestational Diabetes mellitus, it was 21 and in anemia it was 16. In Rh incompatability, it was 12.

Table No. 9.

NO. OF COTYLEDONS	NO. OF PLACENTA	SPECIMEN NO.
10 – 14	13	4, 12, 16, 21, 23…
15 –19	28	2, 3, 5, 8, 13…
20 – 24	43	6, 7, 9, 10, 14…
25 – 29	16	1, 11, 22, 32, 39…

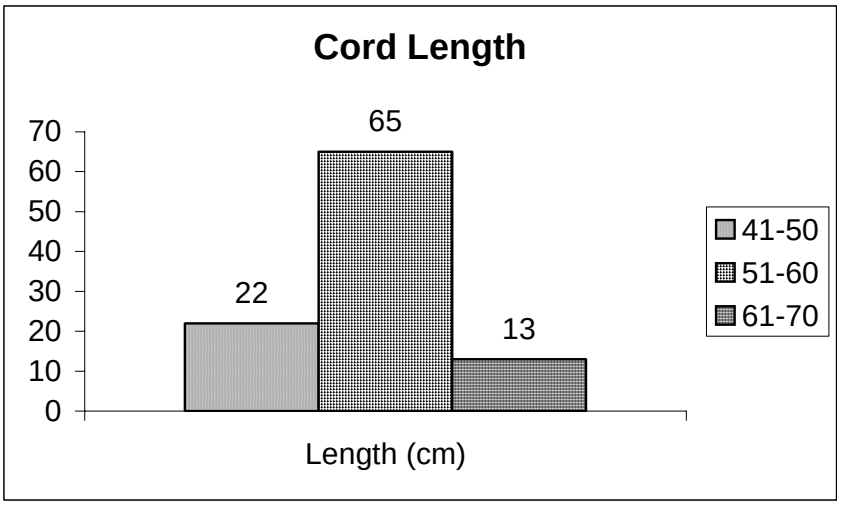


h) CORD LENGTH :

The umbilical cord length ranged from 47-70 cms, average being 54.4 cms.

Table No. 10.

CORD LENGTH (cm)	**NO. OF PLACENTA**	**SPECIMEN NO.**
41 – 50	22	2, 12, 13, 22, 33…
51 – 60	65	1, 3, 4, 5, 6, 7…
61 – 70	13	10, 19, 24, 74, 76…

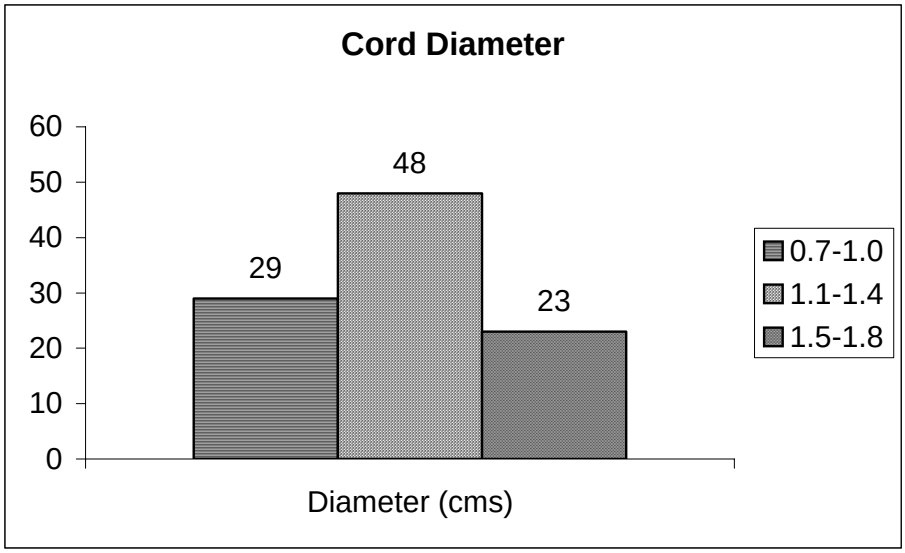


i) DIAMETER OF UMBILICAL CORD :

It was from 0.7 cms to 1.8 cms and the average was 1.24 cms.

Table No. 11.

CORD DIAMETER (cm)	**NO. OF PLACENTA**	**SPECIMEN NO.**
0.7 – 1.0	29	1, 2, 5, 6, 8…
1.1 – 1.4	48	4, 13, 14, 15, 16…
1.5 – 1.8	23	3, 7, 24, 25, 26…

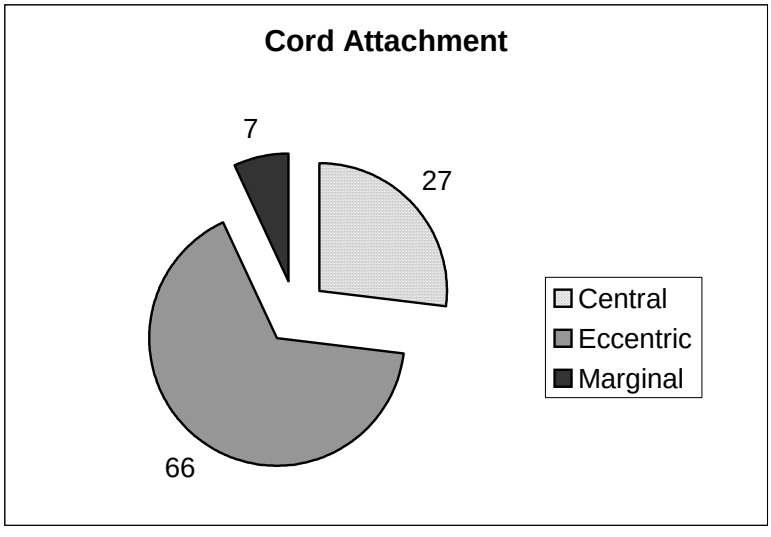


j) CORD ATTACHMENT :

The following types of cord attachment were observed. Most of the cords were attached eccentrically and it was found to be 66. The next common attachment was central, totaling 27. Marginal attachment was seen only in 7 cases.

Table No. 12.

ATTACHMENT	NO. OF PLACENTA	SPECIMEN NO.
Eccentric	66	1, 3, 4, 5, 6…
Central	27	7, 11, 22, 23, 24…
Marginal	7	2, 17, 18, 21, 26, 28, 42



k) UMBILICAL CORD VESSELS :

Normally umbilical cord contains two umbilical arteries and one umbilical vein. In our study also all the umbilical cord contained two umbilical arteries and one umbilical vein.

Table NO. 13.

C/S OF UMBILICAL CORD	NO. OF PLACENTA
2 Umbilical Arteries	100
Single Umbilical Artery	0
Double Umbilical Vein	0

l) KNOTS :

In the present study, false knots were observed in 6 cords. No true knots were observed.

Table No. 14.

KNOTS	NO. OF PLACENTA	SPECIMEN NO.
No Knots	94	1, 3, 4, 5, 6…
False Knots	6	2, 7, 16, 28, 33, 68
True Knots	0	-

m) PLACENTAL CALCIFICATION :

No placental calcification were observed in any of the 100 placenta.

Table No. 15.

PLACENTAL CALCIFICATION	NO. OF PLACENTA
Absent	100
Present	0

DISCUSSION

This study on morphometric analysis of human placenta was carried out as the placenta gives an accurate record of the infant's intrauterine life. From this study, it can be said that a ten minutes careful examination of the placenta reflects the ten months environment from where the child has come.

This is an era of non-invasive techniques and placenta can very much be examined thoroughly during routine ultrasonogram of expectant mothers.

During Ultrasonogram test, due care has to be taken to study the various aspects of placenta and umbilical cord.

In morphometric study of placenta, measurement of its shape, size weight, thickness and the number of cotyledons were measured.

The umbilical cord length, circumference, diameter were measured and its attachment were also noted down.

Details of the mother and child were also obtained for clinical correlation.

This study was done using 100 placentae which were obtained from the labour ward and operation theatres of Rajah Mirasudhar Govt. Hospital.

Of these 100, 85 were from uncomplicated pregnancies and 15 were from factors complicating pregnancy. This 15 cases included 2 cases of gestational Diabetes, 3 cases of Pregnancy Induced Hypertension, 1 case of Rh incompatability, 5 cases of anemia complicating pregnancy, 2 cases of Intra uterine growth retardation and 2 cases of Intra uterine death.

About 93 placentae were circular in shape, while 7 were found to be oval. This finding fits with the normal range seen in other studies.

Three cases of Pregnancy Induced Hypertension were seen. The weight of placentae in these cases respectively 390 gms, 310 gms and 480 gms. The average was 393 gms. There is no difference between this and the average from normal pregnancies. However, these were moderate cases only and severe cases were not encountered.

The average weight of placenta in gestational Diabetes was higher than normal and it was 755 gms. The babies were bigger in all these cases.

The reason for big babies in diabetes remained a matter of controversy. One possible reason cited in literature is dysfunction of Anterior Pituitary gland due to excessive production of diabetogenic and growth producing hormones of Anterior pituitary.

According to Y in Liu Y, the placental weight gradually decreased in prolonged pregnancy as according to them, the placental function is lowered in them. But, in this study no such observation was made.

The placental weight is low in case of mothers who had anemia. Maternal diseases including anemia have their effects on the fetus by altering placental metabolism and transfer mechanisms. The observations made in this regard was consistent with the study of Woohing et al 1976. The average weight of placenta in anemic mothers in this study was 446 gms and 360 gms in previous study of Woodling et al.

The weight of placenta is normally 500 gms. In this study, the minimum weight of placenta was 230 gms & the maximum weight of the placenta was 760 gms and the average was 508.8 gms.

PLACENTAL DIAMETER & THICKNESS :

In this study the placental diameter in case of male babies was found to be 17.9 cms and female babies was 17.5 cms. The term human placenta is 15 to 20 cms according to A.L. Mudaliar. The result of this study falls within the normal range. So also the thickness of placenta at term was 1.88 cms in case of male and 1.71 cms in case of female babies. This parameter also fell within the normal suggested by AL Mudaliar.

The maternal surface of placenta bears cotyledons ranging from 12 to 24 with an average of 18. The average number of cotyledons in present study was 20.

The paucity of lobes in cases of pregnancy induced hypertension and low birth weight as seen by Nordenvall et al was not seen in present study. These cotyleldons were ill defined in cases of anemia complicating pregnancy.

There is no correlation between the number of cotyledons and the sex of the baby either in previous studies or in the present study.

OTHER OBSERVATIONS :

The present study shows translucent membrane in 92 cases, opaque in 2 cases and meconium stained in 6 cases. In one of the 2 opaque membranes, the baby had cord around the neck three times and mildly asphyxiated. It was resuscitated immediately. All the 6 cases of meconium stained membranes were associated with foetal distress.

The fetal surface of placenta was steel blue in colour in most of the cases. Sub-chorionic fibrosis and Tasselations were present in all term placentae. Tasselations are sclerosed vessel below the chorion forming a criss-cross pattern on the foetal surface.

No calcification was noted in any of the 100 placenta. In the present study, retroplacental clots were noticed in 4 cases and all were Abruptio placentae. No congenital anomaly was present in the new born. The average weight of the retroplacental clot was 100 gms.

PLACENTAL WEIGHT IN RELATION TO WEIGHT OF THE BABY :

The ratio of foetal weight to placental weight if 6 : 1, according to Hamilton & Boyd. In the present study, it was 5.98:1 in case of male babies and 6.02:1 in case of female babies. Both were nearer to normal ratio. The foetal weight ratio in cases of gestational diabetes was 5.2, in pregnancy

induced hypertension was 5.8 and in anemia complicating pregnancy 5.8. All these values are also almost nearer to the normal ratio suggested by Hamilton & Boyd.

The otherway to correlate the weight of the baby and placenta is placental coefficient. The placental weight in grams divided by foetal weight in grams is the placental coefficient as defined by Little.

The normal placental coefficient is 0.12 to 0.2. The placental coefficient in the present study for male babies was 0.17 and for female babies also. This placental coefficient in Gestational diabetes was 0.19, Pregnancy Induced Hypertension was 0.17 and in anemia complicating pregnancy was 0.17.

MORPHOLOGICAL PARAMETERS OF THE UMBILICAL CORD :

The umbilical cord is also called the Funiculus Umbilicalis. The minimum cord length in the present study was 47 cms and the maximum was 70 cms. The average cord length was 54.4 cms.

The average length of umbilical cord in African population was 58.4 cms in the study of Nnatu S et al. The length of cord from their study and of

others as well as this established the fact that the cord length are not determined by the racial groups.

This study also observed that there is no significant relation between cord length and parity of mother.

The diameter of the cord varied from 0.7 cms to 1.8 cms in the present study. The average was 1.24 cms.

Out of 100 cases, Eccentric type of cord insertion was seen in 66 cases, central type of cord insertion in 27, marginal insertion in 7. Velamentous type and Battledore type of insertion were not encountered.

The mode of umbilical cord insertion has no significant effect on compartmental volume of placental parenchyma.

The cord insertion is visualized in more than half of the cases in Ultrasonography in clinical practice and the knowledge is useful for planning obstetrical management.

The calculated length of umbilical cord is almost equivalent to actual length measured after delivery.

The thickness (circumference) of the umbilical cord includes the umbilical vessels and the connective tissue called as Whartons Jelly. All these are surrounded by a sheath formed by the amnion. For a short distance from its junction with anterior abdominal wall, the cord is covered by an epithelial tissue.

Marginal cord insertion is usually related to bilobate placenta. Major fetal anomalies with normal Karyotype are found in Velamentous type.

The cord insertion is probably the effect of biological variations during normal placental and fetal development. Marginal cord insertion may evolve into velamentous insertion as pregnancy progresses.

In the present study, no true knot (nodi veri) was observed. However, false knots (nodi spurii) was observed in 6% of cases. They are formed by umbilical Vein twisting around the umbilical artery, causing a localized thickening of Wharton's Jelly and they lack any clinical significance.

In the present study, Whartons jelly was present in all the cases. If absent, they are associated with other congenital anomalies.

In the present study, no anomaly of umbilical vessels were noticed.

SUMMARY

The summary of the study of morphology & morphometric analysis of placenta is as follows:

A total of 100 specimens of placenta and umbilical cord were studied in the present study.

The placenta were derived from 53 primigravida and 47 multigravida. 85 were from uncomplicated cases and 15 were from factors complicating pregnancy. Out of 100 specimens, 52 were from male, 48 from female.

Out of 100 specimens studied, 93 were circular, 7 were oval.

The weight of the placenta ranges from 230 gms to 680 gms in case of female with an average of 512.71 gms & 250 gms to 760 gms with an average of 504.81 gms in case of male children.

The diameter of the placenta in the present study ranges from 14 cms to 22 cms, average being 17.6 cms.

The placental thickness ranges from 1.1 cms to 2.5 cms, with an average of being about 1.8 cms.

The ratio between the foetal weight and placental weight in case of male was 5.98 and in case of female was 6.02.

The placental coefficient in the present study is 0.17 both in male and female.

The average number of cotyledons was 20.

Subchorionic fibrosis and Tasselations seen in all placentae.

No placental calcification seen.

Retroplacental clots seen in 4 cases and all of them were associated with Abruptio placentae.

The average length of the umbilical cord was 54.4 cms.

Present study showed eccentric type of cord insertion in 66, central type in 27, Marginal type in 7.

False knots noticed in 6 cases.

Diameter of the umbilical cord on an average was 1.24 cms. Whartons Jelly present in all specimens.

CONCLUSIONS

Morphological studies of placenta in high risk group and subsequent cytogenetic analysis will prove presence or absence of chromosomal foetal malformations in terms of 8 – 24 weeks when termination is possible. Morphometry is an indirect and non-invasive approach to the study of the physiology and physiopathology of gestation in the human. Morphometry is a good adjuvant to histopathology.

Gross examination was more informative, though microscopic examination would have been necessary.

Triage is the sorting and allocation of treatment according to a system of priorities in order to maximize treatment. Placental triage promptly after delivery of the placenta with documentation of the findings in the medical record only takes a few minutes, and allows for the identification of abnormal placentae to be submitted for detailed gross and microscopic examination. It requires familiarity with normal gross placental anatomy.

Our study concludes with a recommendation that a systematic procedure has to be adopted and documentation for comprehensive examination of the placental disk as a whole, the umbilical cord, the extra placental membranes, the fetal surface, the maternal surface and the parenchyma. Otherwise, the placenta is dumped into the dustbin with all its useful information.

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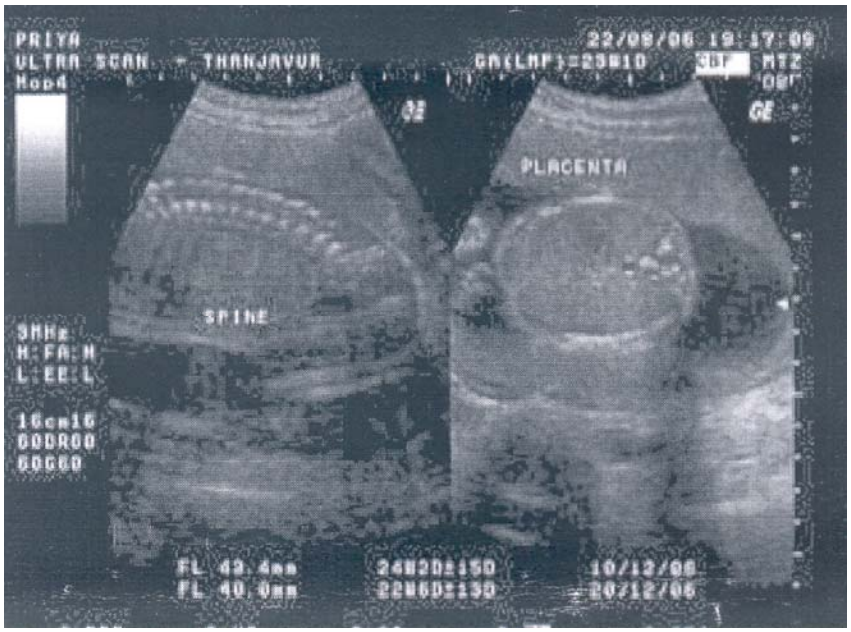
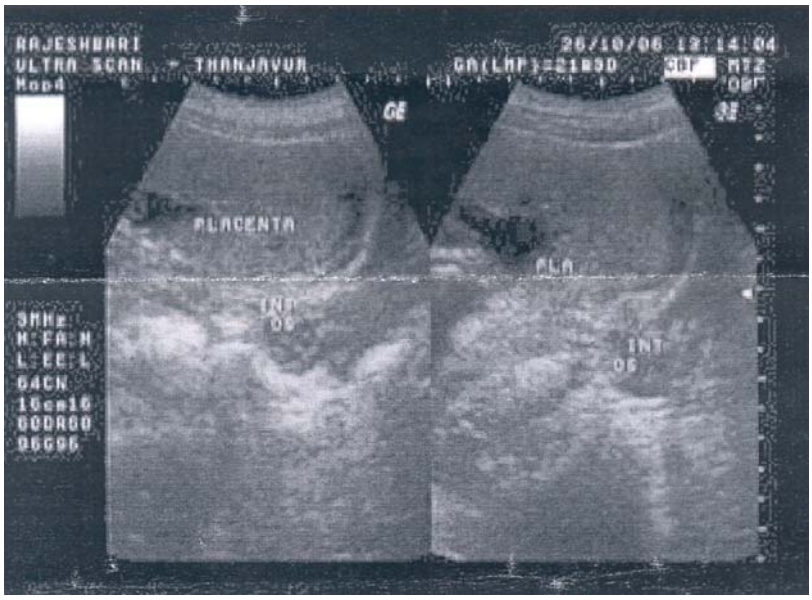
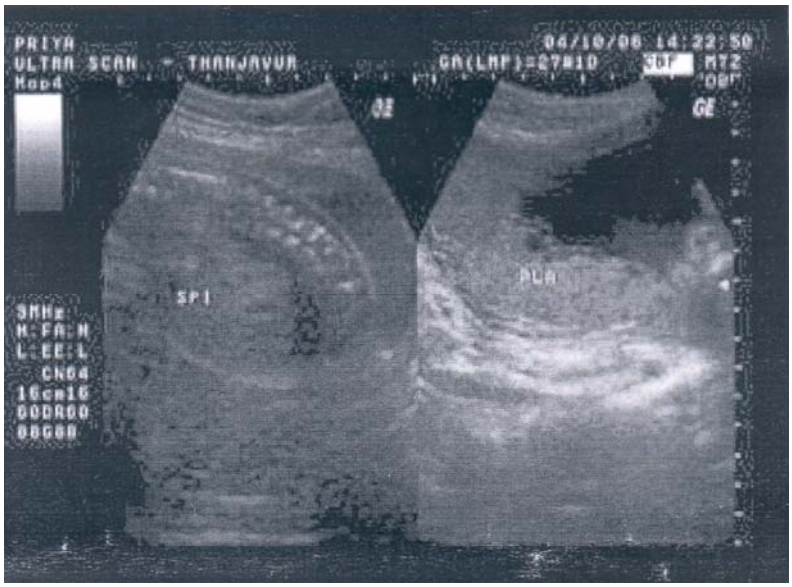


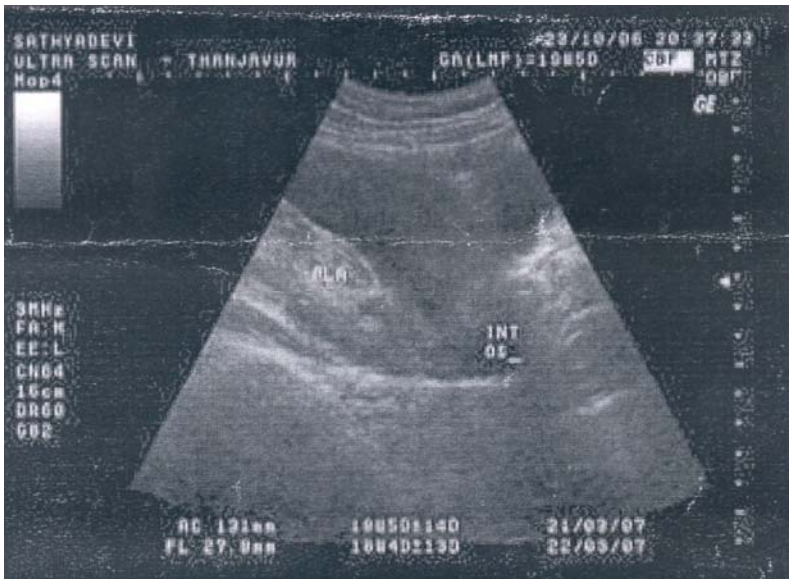
Placenta : Fundal Anterior
35 mm Away from the internal os.



Placenta : Low Lying
Completely covering the internal os.



Placenta : Fundal Posterior
Well away from the internal os.



Placenta : Fundal Posterior,
Touching the internal os.

S. No	Name		Mother			Baby		Placenta				Umbilical cold		
			Age	Ob. Code	Cy Complicatry	Sex	Weight	Wt.	Diam	Thickness	No. of Coty.	Length	Diam	Attachment
1.	Mrs. Usha	41006	28	Primi	-	F	3.0	500	18	1.4	29	58	0.8	Eccentric
2.	Mrs. Devi Chitra	41109	26	Primi	-	M	2.1	350	14	1.2	19	50	1.0	Marginal
3.	Mrs. Sulfana	41197	20	Primi	-	F	3.2	580	15	1.8	15	52	1.6	Eccentric
4.	Mrs. Radhika	41266	22	Primi	RH incomp	M	2.4	410	15	2.3	12	57	1.1	Eccentric
5.	Mrs. Priya	41120	24	Primi	-	F	3.1	510	17	2.2	18	51	0.9	Eccentric
6.	Mrs. Sasiseela	41159	23	Primi	-	F	2.6	425	17	1.4	22	53	0.9	Eccentric
7.	Mrs. Sumathi	41287	22	Primi	Diabetes	M	4.2	760	15	2.2	20	52	1.8	Central
8.	Mrs. Juliet	41105	33	$G_2P_oL_o$	-	F	3.3	530	14	2.3	16	53	0.8	Eccentric
9.	Mrs.Rajakumari	41053	25	$G_2P_1L_1$	-	M	3.1	510	19	1.7	20	56	0.8	Eccentric
10.	Mrs. Eswari	41290	25	Primi	-	M	3.2	540	18	1.8	24	62	0.8	Eccentric
11.	Mrs. Rupa	41117	23	$G_2P_1L_1$	-	F	3.6	600	17	1.6	28	51	0.7	Central
12.	Mrs. Udayakumari	41105	26	Prim	PIH	F	2.3	390	17	1.8	12	49	1.0	Eccentric
13.	Mrs.NajumuNisha	41128	24	$G_3P_1L_1$	-	M	2.4	400	16	1.2	15	50	1.1	Eccentric
14.	Mrs.Vanitha	41285	24	$G_2P_1L_1$	-	F	3.1	500	19	1.7	24	54	1.2	Eccentric
15.	Mrs.Senthamilselvi	41125	26	Primi	-	M	2.9	490	18	2.3	17	51	1.4	Eccentric
16.	Mrs. Pandiammal	41114	20	Primi	-	M	2.8	480	16	2.2	12	52	1.3	Eccentric
17.	Mrs. Jeya	41116	23	$G_2P_1L_1$	-	F	3.1	510	16	1.6	20	52	1.4	Marginal
18.	Mrs. Suryabanu	40352	25	Primi	Diabetes	M	3.7	750	17	2.0	21	51	1.0	Marginal

19.	Mrs. Anbu	40236	31	$G_2P_1L_1$	-	F	3.1	510	15	1.7	22	62	0.8	Eccentric
20.	Mrs. Vasanthi	40873	33	$G_3P_2L_2$	-	F	3.3	550	19	1.9	24	60	1.2	Eccentric
21.	Mrs. Parvathi	42034	28	$G_2P_1L_1$	-	M	2.5	400	16.5	1.7	12	53	1.4	Marginal
22.	Mrs.Krishnaveni	41203	27	$G_1L_1L_0$	PIH	M	2.8	480	17	2.0	26	49	1.0	Central
23.	Mrs. Papathi	41508	21	Primi	-	M	2.4	400	14	1.4	10	52	1.1	Central
24.	Mrs. Parimala	41630	22	Primi	-	F	3.8	610	18	1.7	18	63	1.8	Central
25.	Mrs. Hema	41622	24	$G_3P_1L_1$	-	F	3.2	530	16	1.8	20	58	1.7	Central
26.	Mrs. Kavitha	40965	26	$G_2P_1L_0$	-	M	3.2	530	19	1.9	20	60	1.6	Marginal
27.	Mrs. Lakshmi	40774	34	$G_4P_3L_3$	-	F	4.0	680	15	1.6	20	54	1.3	Central
28.	Mrs. Kalpana	41007	20	Primi	-	M	3.6	570	19	1.8	20	51	1.2	Marginal
29.	Mrs. Mala	41554	23	Primi	-	M	3.0	500	18.5	2.5	22	50	1.3	Eccentric
30.	Mrs. Karpagam	41587	25	$G_2P_1L_1$	-	F	3.2	520	17	2.0	24	53	1.4	Eccentric
31.	Mrs.Leela	42052	21	Primi	-	F	3.5	580	20	2.2	23	52	1.5	Eccentric
32.	Mrs. Mookambigai	42047	27	$G_2P_1L_1$	-	M	3.5	570	18	1.8	28	57	0.9	Eccentric
33.	Mrs.Elizabeth	40489	20	Primi	-	F	2.9	490	15	1.7	18	49	1.0	Central
34.	Mrs.Aadhiammal	4050	18	Primi	PIH	M	1.8	310	16	2.3	15	47	0.7	Eccentric
35.	Mrs.Lalitha	42158	17	Prim	-	F	2.7	420	16	1.6	18	52	0.9	Eccentric
36.	Mrs. Poornima	40227	19	Prim	-	F	2.8	460	18	2.1	18	54	1.2	Central
37.	Mrs. Mohana	41587	28	$G_3P_0L_0$	IUD	M	1.5	250	17	1.9	15	51	1.5	Central
38.	Mrs. Amudha	40118	20	Prim	-	M	2.7	450	20.5	1.3	17	53	1.3	Eccentric
39.	Mrs. Vijaya	41196	21	Prim	-	F	3.6	590	19	2.4	28	60	0.8	Central

40.	Mrs. Mala	40334	24	$G_2P_1L_1$	-	M	3.75	620	19	2.2	26	55	1.4	Eccentric
41.	Mrs. Saral	41388	22	Prim	-	F	3.2	520	15	1.6	20	52	1.6	Eccentric
42.	Mrs.Arokya Mary	41391	29	$G_3P_2L_2$	-	M	3.1	510	20	2.3	19	51	1.6	Marginal
43.	Mrs. Valli	41587	23	G_2P_2	-	M	2.7	450	18	1.9	16	50	1.7	Eccentric
44.	Mrs. Selvi	41552	21	Primi	-	F	3.5	580	17	1.7	20	48	1.3	Eccentric
45.	Mrs. Jeeva	42087	19	Prim	-	M	2.6	420	18	1.9	11	49	1.4	Eccentric
46.	Mrs. Kalaiselvi	41212	23	Primi	-	F	2.9	480	15	1.7	16	47	1.2	Eccentric
47.	Mrs.Kanimozhi	41141	25	$G_2P_1L_1$	IUGR	M	2.2	370	19	1.4	12	51	1.1	Eccentric
48.	Mrs. Vendha	41215	31	$G_3P_2L_2$	-	F	2.8	490	16	1.7	12	52	1.8	Eccentric
49.	Mrs. Chithra	40936	24	Primi	IUGR	F	2.7	440	16	1.5	16	47	1.0	Eccentric
50.	Mrs. Rekha	41108	28	Primi	-	M	3.2	530	18	1.9	24	49	1.5	Eccentric
51.	Mrs. Shanthi	41230	25	Prim	-	M	3.25	540	14	1.8	19	50	1.1	Eccentric
52.	Mrs.Shobhana	41222	24	$G_2P_0L_0$	IUD	F	1.4	230	19.5	1.2	12	55	0.8	Eccentric
53.	Mrs. Vanitha	41069	26	$G_2P_1L_1$	-	F	2.7	460	16	1.4	20	53	0.7	Central
54.	Mrs.Mareeswari	41152	32	$G_3P_2L_2$	Anaemia	F	2.8	490	20	1.2	20	50	0.9	Central
55.	Mrs. Megala	41699	28	$G_2P_1L_1$		F	3.2	510	22	1.7	23	49	0.8	Central
56.	Mrs. Sankari	41281	20	Primi	-	M	3.7	610	19	2.0	18	53	0.9	Eccentric
57.	Mrs.Maneeswari	41994	22	Primi	-	F	3.6	590	17	1.1	18	58	1.2	Central
58.	Mrs. Kala	41475	27	$G_2P_1L_1$		F	2.9	490	16	1.3	13	60	0.8	Central
59.	Mrs.Devi	40551	32	$G_3P_2L_2$	-	F	3.8	630	16	1.2	26	57	1.6	Central
60	Mrs.Santha	42334	30	$G_2P_1L_1$		M	3.2	540	18	1.4	20	49	1.8	Eccentric

61.	Mrs.Jasmine	41274	22	Primi		M	2.9	490	22	1.4	22	51	1.8	Central
62.	Mrs,Mahaswari	41076	20	Primi		F	3.2	535	18	1.3	15	52	1.6	Central
63.	Mrs.Radha	40376	24	$G_2P_1L_1$	-	M	4.0	670	19	2.1	26	56	0.9	Central
64.	Mrs.Padma	41980	26	$G_2P_1L_1$	-	M	2.8	450	17	1.7	14	58	1.8	Eccentric
65.	Mrs.Latha	41877	28	$G_2P_1L_1$	-	M	3.5	580	18	1.8	18	54	1.6	Eccentric
66.	Mrs.Sudha	40246	24	Primi	-	M	3.2	540	20	1.4	24	54	1.2	Eccentric
67.	Mrs.Jeyanthi	41756	23	Primi	-	M	3.8	680	19	1.2	22	52	1.1	Central
68.	Mrs.Revathy	42345	24	$G_2P_1L_1$	-	F	2.8	450	22	1.1	15	48	1.4	Eccentric
69.	Mrs.Santhi	40658	25	$G_2P_1L_1$	-	M	3.2	530	18	1.9	18	48	1.8	Eccentric
70.	Mrs.Maragatham	40924	23	Primi	-	F	3.7	610	19	2.1	20	50	0.9	Central
71.	Mrs.Pushpam	41839	24	Primi	-	F	2.9	490	15	1.7	15	53	1.2	Central
72.	Mrs.Gnanam	40629	16	Primi	Anaemia	M	2.2	380	15	1.5	12	49	1.3	Eccentric
73.	Mrs.Bagavathy	40723	33	$G_3P_2L_1$	-	F	3.7	620	20	2.2	20	56	1.4	Eccentric
74.	Mrs.Eshwari	41237	20	Primi	-	M	3.4	570	22	2.4	22	63	1.6	Eccentric
75.	Mrs.Nayaki	40275	18	Primi	Anaemia	M	2.7	460	18	1.9	20	54	1.5	Central
76.	Mrs.Pandeeswari	41387	28	$G_4P_3L_2$	Anaemia	F	2.8	460	16	1.8	18	68	1.7	Eccentric
77.	Mrs.Muniammal	41397	22	Primi	Anaemia	F	2.4	440	15	1.7	10	52	1.4	Eccentric
78.	Mrs.Annammal	41998	23	Primi	-	M	3.2	520	20	2.2	22	57	1.1	Eccentric
79.	Mrs.Malliga	40098	24	$G_2P_1L_1$	-	F	3.5	580	19	2.1	22	55	1.8	Eccentric
80.	Mrs.Jothi	40076	21	Primi	-	M	3.7	580	18	2.0	18	62	1.3	Eccentric
81.	Mrs. Bhuvaneswari	40078	20	Primi	-	M	2.8	480	17	1.9	20	59	0.9	Eccentric

82.	Mrs. Fathima	40079	23	$G_2P_1L_1$	-	M	3.4	540	19	2.1	29	51	1.4	Central
83.	Mrs. Sridevi	40065	29	$G_2P_1L_1$	-	M	3.4	540	19	2.1	29	51	1.4	Eccentric
84.	Mrs.Patmavathi	40069	18	Primi	-	M	3.5	560	16	1.8	20	53	1.2	Eccentric
85.	Mrs.Vimala	40062	20	Primi	-	F	2.8	490	15	1.7	26	52	1.1	Eccentric
86.	Mrs. Manimala	40125	19	Primi	-	F	2.7	460	15	1.5	20	50	1	Eccentric
87.	Mrs.Ramjan Bevi	40352	25	$G_2P_1L_1$	-	F	3.4	540	17	2	25	55	1.2	Eccentric
88.	Mrs.Manimegalai	40362	25	$G_2P_1L_1$	-	F	2.8	470	20	2.2	27	55	1.2	Eccentric
89.	Mrs.Chellama	41256	25	$G_3P_2L_2$	-	M	2.9	490	19	2.4	22	62	1.3	Eccentric
90.	Mrs.Amala	42352	34	$G_3P_2L_2$	-	F	2.7	440	14	2.0	19	65	1.2	Eccentric
91.	Mrs. Durga	42355	28	$G_2P_1L_1$	-	M	3.2	530	17	2.0	20	55	1.1	Central
92.	Mrs. Sailaja	42218	27	$G_2P_1L_1$	-	M	3.0	500	17	1.8	22	58	1.4	Eccentric
93.	Mrs.Sabeena	40258	36	$G_3P_2L_2$	-	M	3.0	500	19	2.0	24	60	1.4	Eccentric
94.	Mrs.Saradha	41852	28	$G_2P_1L_1$	-	M	2.7	440	21	2.4	24	65	1.4	Eccentric
95.	Mrs.Kumudhavalli	41864	20	Primi	-	F	3.1	520	15	2.0	20	65	1.2	Central
96.	Mrs.Anandaselvi	40559	19	Primi	-	M	3.2	530	18	1.8	26	70	1.0	Eccentric
97.	Mrs.Jeyalakshmi	40684	22	Primi	-	F	3.4	590	21	1.6	22	57	1.2	Eccentric
98.	Mrs.Kurnnammal	41338	23	$G_2P_1L_1$	-	M	2.9	480	22	1.6	27	56	1.1	Eccentric
99.	Mrs.Annathai	40521	20	Primi	-	M	2.8	470	18	2.0	26	62	1.4	Eccentric
100	Mrs.Priya	40886	22	Primi	-	F	3.2	520	17	1.5	20	65	1.2	Eccentric